Rapid and reversible formation of spine head filopodia in response to muscarinic receptor activation in CA1 pyramidal cells

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Abstract: A key feature at excitatory synapses is the remodelling of dendritic spines, which in conjunction with receptor trafficking modifies the efficacy of neurotransmission. Here we investigated whether activation of cholinergic receptors, which can modulate synaptic plasticity, also mediates changes in dendritic spine structure. Using confocal time-lapse microscopy in mouse slice cultures we found that brief activation of muscarinic receptors induced the emergence of fine filopodia from spine heads in all CA1 pyramidal cells examined. This response was widespread occurring in 48% of imaged spines, appeared within minutes, was reversible, and was blocked by atropine. Electron microscopic analyses showed that the spine head filopodia (SHFs) extend along the presynaptic bouton. In addition, the decay time of miniature EPSCs was longer after application of the muscarinic acetylcholine receptor agonist methacholine (MCh). Both morphological and electrophysiological changes were reduced by preventing microtubule polymerization with nocodazole. This extension of SHFs during cholinergic receptor activation represents a novel structural form of subspine plasticity that may regulate synaptic properties by fine-tuning interactions between presynaptic boutons and dendritic spines.

DOI: https://doi.org/10.1113/jphysiol.2010.204446

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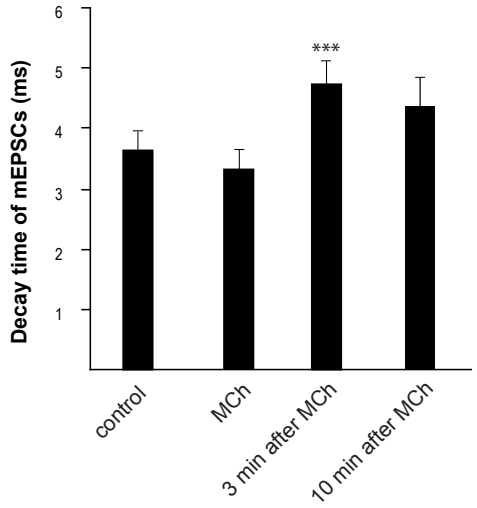
Decay time of mEPSCs (ms)
6
5
4
3
2
1
***
control
MCh
3 min after MCh
10 min after MCh